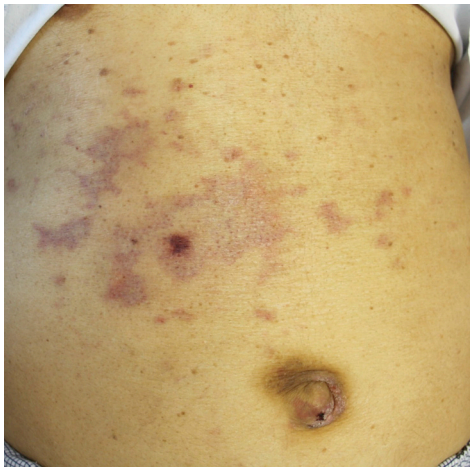

Fig 1. Supraumbilical cutaneous lesions 3 days after transcatheter arterial chemoembolization. Violaceous indurated plaques over the navel.

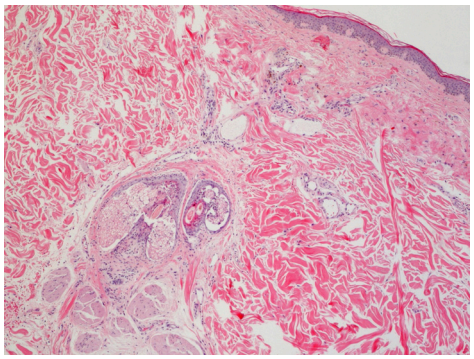

Fig 2. Biopsy showing, in the dermis, focal necrosis of the sebaceous gland and presence of ghost cells with loss of hematoxylin stain in the pilosebaceous unit. Note that dermal collagen does not show fibrosis or sclerosis. (Hematoxylin-eosin stain; original magnification: ×40.)

due to the small size, low velocity of arterial flow, or L-shaped angle of the HFA.[1]

The skin lesions consist of violaceous, indurated, and painful plaques in the supraumbilical area that can evolve to complete necrosis of the abdominal wall.[3] Histology is nonspecific, displaying sclerosis with vascular thrombosis and focal fat necrosis.[3]

Treatment consists of local application of cold packs, topical antibiotics, and surgical debridement if necessary. In 1 case, oral pentoxifylline was used with good results.[3]

This complication might be avoided by prophylactic embolization of the proximal portion of the HFA using microcoils, thus preventing the flow of the chemotherapeutic agent to this artery.[3,4] Because the abdominal wall is also irrigated by branches of the superior and inferior epigastric arteries, which communicate with the HFA, embolization would not cause ischemic lesions on the skin.[4] Because the HFA is a small caliber artery that is sometimes difficult to access, Wang et al[5] proposed the prophylactic use of ice packs on those patients whose HFA cannot be embolized.

We think it is important to acknowledge the risk of developing supraumbilical cutaneous lesions after TACE. Although it is a rare complication of this procedure, its frequency will probably grow in the future due to the increased use of TACE in the treatment of unresectable liver tumors.

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Merkel cell carcinoma presenting as right leg edema in a multiple myeloma patient

To the Editor: A 61-year-old man with intractable vomiting, fatigue, and acute renal failure was accurately diagnosed with stage III multiple myeloma as

Fig 1. Merkel cell carcinoma. Computed tomography–guided needle biopsy of the nodal mass.

Fig 2. Merkel cell carcinoma. Cutaneous in-transit metastases.

the initial neoplasm. He was treated with lenalidomide, dexamethasone, and bortezomib with an excellent response. Four months after treatment completion, he presented with persistent right lower extremity edema and pain. Deep vein thrombosis and renal artery stenosis were excluded with Doppler ultrasound. Physical examination revealed pitting edema to the knee with dusky erythema and scaling; laboratory investigations were significant for kidney failure. A computed tomography (CT) scan of the abdomen and pelvis revealed a 14- × 7-cm retroperitoneal nodal mass obstructing the ureters and bilateral hydronephrosis. A CT-guided biopsy revealed Merkel cell carcinoma (MCC) (Fig 1). No cutaneous lesions were evident at that time. The patient received external beam radiation therapy for the mass, which alleviated the edema and pain.

Shortly thereafter he returned with painful, firm, erythematous nodules on his distal right leg, which demonstrated metastatic MCC on needle biopsy. He began a palliative regimen of carboplatin and etoposide, which resulted in pancytopenia and sepsis. The lesions recurred throughout his leg, thigh, and foot 1 month later with ulceration and bleeding (Fig 2). He subsequently began gemcitabine, which resulted in anemia requiring transfusions and metastatic progression. Chemotherapy was changed to temozolomide. Six months later, widely disseminated cutaneous lesions involved the left lower extremity, abdomen, and chest. Chemotherapy was discontinued in favor of palliative measures, and the patient died of disease progression 3 months later.

MCC is a rare, highly aggressive neuroendocrine malignancy with a high rate of local recurrence and metastasis, and the diagnosis carries a poor prognosis. Increasing evidence has demonstrated a relationship between MCC and impaired immunity, whether iatrogenic after organ transplantation, acquired due to human immunodeficiency virus, or intrinsic due to malignancy.[1] This association led to the discovery of the Merkel cell polyomavirus and its detection in the majority of MCC tumors, revealing an increased frequency of virus reactivation in immunocompromised patients.[2] Recent case reports have suggested that primary cancers, especially chronic lymphocytic leukemia, non-Hodgkin lymphoma, and multiple myeloma, may predispose patients to development of MCC. Howard et al[3] demonstrated a greater than 3-fold increased risk of primary MCC development after multiple myeloma diagnosis with a median latency of 3.4 years.

This case of MCC presenting with acute renal failure and impaired lower extremity venous drainage causing edema illustrates that the diagnosis of MCC may have been confounded by the patient's preexisting malignancy and residual kidney impairment due to light chain deposition. We also believe that persistent lymphedema of the right leg, which was likely the primary site of disease, masked the detection of this second primary malignancy. When treating immunocompromised patients, especially those with lymphohematopoietic malignancies, vigilance for the development of MCC should be considered, in that early detection can greatly impact prognosis and survival. This case reemphasizes that cutaneous malignancy risk assessment should be performed in the initial stages of management for every immunocompromised patient.

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Photosensitive lichenoid drug eruption to capecitabine

To the Editor: A 75-year-old Caucasian woman undergoing capecitabine treatment for metastatic breast cancer presented to the dermatology clinic for evaluation of a pruritic eruption on the sun-exposed areas of her body that began 2 months after initiation of capecitabine treatment. The patient reported receiving ample sun exposure during the summer months when she noted the onset of the eruption. She also noted fingernail changes over the same time frame. Her other medications included prochlorperazine maleate 10 mg, hydrocodone/acetaminophen 5 mg/325 mg, and denosumab 120 mg subcutaneously every 4 weeks. Examination revealed numerous violaceous, flat-topped papules on extensor forearms, dorsal hands, and anterior legs (Fig 1). Scaling and erythema was observed in the periungual areas with marked subungual hyperkeratosis. Diffuse erythema was also noted of the palmar and plantar surfaces. Examination of the oral cavity was unremarkable. Fingernail clipping for histopathologic and microbiologic evaluation of fungus was negative. Punch biopsy of the skin of her right dorsal hand demonstrated a lichenoid interface dermatitis with compact orthokeratosis of the stratum corneum, multiple scattered melanophages, and a few eosinophils in the dermis. The clinical and histologic features were consistent with a diagnosis of photosensitive lichenoid drug eruption secondary to capecitabine treatment. The patient was prescribed topical clobetasol propionate 0.05% ointment and systemic hydroxyzine, and was recommended adherence to sun protection strategies. With these measures, she noted a marked improvement in her eruption without any changes in her chemotherapeutic regimen.

Capecitabine (Xeloda) is an oral prodrug of fluorouracil (5-FU) used in the treatment of advanced colorectal, esophageal, laryngeal, and metastatic breast cancer.[1] While 5-FU has been used in the past for many solid tumors, it must be administered intravenously. As an oral prodrug of 5-FU, capecitabine is easier to administer and more convenient for the patient; as such, its use has increased in treating these patients.[2] Some reports have suggested a link between capecitabine use and the development of a lichenoid photosensitive eruption.[3] Other cutaneous side effects have also been reported, the most common of which is palmar-plantar erythrodysesthesia, or hand-foot syndrome, which occurs in as many as 50% of those treated with capecitabine.[4] This condition causes tingling in the palms and soles that may progress to burning pain 3 to 4 days later with swelling, erythema, and possible skin ulceration.[3] In addition, reports have linked the use of capecitabine to other cutaneous presentations, including oral lichenoid stomatitis,[1] subacute cutaneous lupus erythematosus,[2] and other dermatologic conditions (Table I).

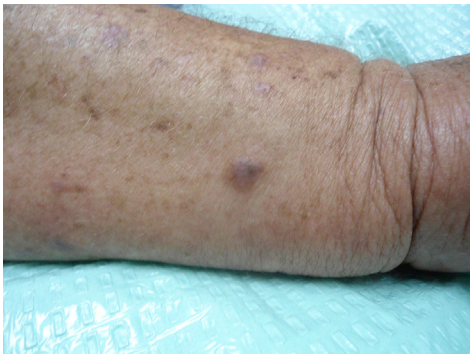

Fig 1. Photosensitive capecitabine drug eruption. Violaceous papules on the extensor forearm.

The reaction in our patient was unique because of the simultaneous presentation of a photosensitive lichenoid eruption, diffuse palmoplantar erythema, and ungual toxicity, as evidenced by the periungual erythema and subungual hyperkeratosis. With the increasing use of capecitabine in chemotherapeutic treatment for cancer, dermatologists should be aware of its potential cutaneous side effects.

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